Gestational Thrombocytopenia Complicated with Macrosomia, Failure to Progress in Active Labor, and Postpartum Hemorrhage

Shih-Peng Mao[1,2], Cheng-Chang Chang[2], Shu-Ying Chen[3], Hung-Chung Lai[2]*

[1]*Division of Obstetrics and Gynecology, Armed Force Kaohsiung General Hospital, Kaohsiung,*
[2]*Department of Obstetrics and Gynecology, Tri-Service General Hospital, Taipei, and* [3]*Basic Medical Science, Department of Nursing, Hung-Kuang University, Taichung, Taiwan.*

SUMMARY

Objective: Gestational thrombocytopenia is a rare event, and the etiology is unknown. Generally, there is no need for intervention because of the absence of coagulopathy. However, when complicated with other obstetric conditions, care should be taken to prevent a dangerous cascade. Here, we present a patient with severe gestational thrombocytopenia complicated with macrosomia, failure to progress in active labor, and severe postpartum hemorrhage after cesarean section.

Case Report: A 25-year-old, gravida 4, para 0, patient from our antenatal clinic developed thrombocytopenia with advancing gestation. Severe thrombocytopenia (platelets, 53×10^9/L) and suspected macrosomia were noted at 39 3/7 weeks of gestation. Induction of labor was conducted for a planned vaginal delivery, but the active labor failed to progress. A cesarean section was performed instead, resulting in immediate postpartum hemorrhage due to uterine atony. Uterine massage, direct compression, and 10 IU of oxytocin (Piton-S, 10 IU/mL; PT Organon, Indonesia) improved uterine contraction only temporarily. Misoprostol was administered rectally. The patient was given a transfusion of packed red blood cells and single-donor platelets. Her condition stabilized after intensive intervention.

Conclusion: Gestational thrombocytopenia does not usually require treatment if there is no bleeding tendency. However, when other bleeding complications are present, it may exacerbate coagulopathy and exhaust the platelet reserve, thereby worsening the condition. If surgical intervention cannot be avoided, blood and platelet transfusion before a cesarean delivery is highly recommended in severe gestational thrombocytopenia. [*Taiwan J Obstet Gynecol* 2007;46(2):177–179]

Key Words: cesarean section, gestational thrombocytopenia, postpartum hemorrhage, thrombocytopenia, uterine atony

Introduction

The normal platelet count in non-pregnant women is $150–400 \times 10^9$/L. Some studies reported that the platelet count decreases by an average of 10% during the third trimester as a result of hemodilution or accelerated destruction leading to younger and larger platelets [1]. Hyperdestruction of platelets may occur in pregnancy, with a consequent decrease in platelet life span [2]. The incidence of thrombocytopenia in pregnant women is approximately 10%. Most thrombocytopenias in pregnancy will be due either to gestational thrombocytopenia (74%) or to hypertensive disorders of pregnancy (21%). Approximately 4% are associated with immune disorders of pregnancy, including idiopathic thrombocytopenia purpura. The remaining 2% comprises a large

Correspondence to: Dr Hung-Chung Lai, Department of Obstetrics and Gynecology, Tri-Service General Hospital, 325, Section 2, Cheng-Gong Road, Neihu, Taipei 114, Taiwan.
E-mail: msp1107@gmail.com
Accepted: January 5, 2007

number of other disorders that range from diffuse intravascular coagulation, and thrombotic thrombocytopenic purpura to acute fatty liver, HELLP syndrome, and antiphospholipid syndrome, among others. Most thrombocytopenias encountered in pregnancy are etiologically unknown [3]. Gestational thrombocytopenia is the most common cause of thrombocytopenia during pregnancy and is usually without severe complications [4]. This article reports a patient with postpartum hemorrhage (PPH) and coexisting severe gestational thrombocytopenia.

Case Report

A 25-year-old woman, gravida 4, para 0, visited our clinic regularly during her pregnancy. Her antenatal care was uneventful until the 39th week of gestation when she was admitted for induction of labor because of possible macrosomia (estimated fetal body weight, 3,913 g) and a ripen cervix (Bishop's score, 4). The initial investigation revealed a reactive nonstress test and absence of uterine contraction. The laboratory workup revealed a normal hemogram, except for severe thrombocytopenia (platelets, 53 × 10^9/L), making vaginal delivery a mandatory choice. The prothrombin and partial thromboplastin time were both within normal limits. Labor was induced with vaginal misoprostol 50 μg. The uterus reached regular uterine contractions 1 hour later with a good beat-to-beat variation. However, the progress of labor was unsatisfactory, and a cesarean section was performed because of failure to progress in active labor. A male baby was delivered, and his body weight was 4,190 g. The Apgar scores were 8 and 9 at 1 and 5 minutes, respectively.

Unfortunately, uterine atony with immediate postpartum hemorrage (PPH) developed during the operation. Uterine massage, direct uterine compression, intramuscular injection of methylergonovine maleate 0.2 mg (Neo-Ergo, 0.2 mg/mL; Oriental, Taiwan) and intravenous oxytocin 10 IU (Piton-S, 10 IU/mL; PT Organon, Indonesia) were administered during the operation. The uterine tone improved temporarily. After she returned to the ward, massive uterine bleeding (estimated amount, > 1,500 mL) occured again. Misoprostol 400 μg as a rectal suppository was administered. The hemogram showed extremely severe thrombocytopenia (platelets, 23 × 10^9/L) and severe anemia (hemoglobin, 4.3 g/dL). Transfusions of packed red blood cells (4 units) and single-donor platelets (2 units) were given immediately. After intensive intervention, the patient's condition stabilized gradually. She was discharged 5 days after childbirth. The change in her platelet count is shown in the Figure.

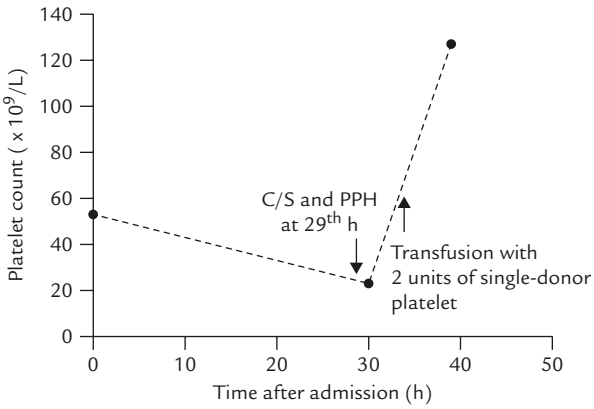

Figure. The change of platelet count and hospital course of the patient with thrombocytopenia and postpartum hemorrhage. C/S = cesarean section; PPH = postpartum hemorrhage.

Gestational thrombocytopenia, which accounts for approximately 74% of cases of maternal thrombocytopenia, usually develops in the third trimester, with a mild to moderate decrease at delivery. The possible causes of thrombocytopenia in pregnancy can be divided to three categories: platelet destruction or consumption, splenic sequestration of platelets, and failure of platelet production in the bone marrow.

Discussion

Gestational thrombocytopenia, like autoimmune thrombocytopenia, is classified into the first category [5]. The actual cause is unknown; however, there is evidence of physiologic platelet activation during pregnancy at the site of placental circulation, leading to shortening of platelet lifespan [2]. Gestational thrombocytopenia, which can hardly be distinguished from autoimmune thrombocytopenia antenatally, is a diagnosis of exclusion and can only be suggested by a late decrease in platelet count in a patient without history of this disorder. The patient presented here did not have any past history of autoimmune diseases, and her platelet count returned to normal shortly after the delivery; thus, the diagnosis was most likely to be gestational thrombocytopenia.

Usually, treatments or invasive approaches to fetal monitoring are not necessary. In this case, the platelet count showed moderate thrombocytopenia (53 × 10^9/L). Initially, the patient attempted a vaginal birth. However, failure to progress in active labor and macrosomia, which are commonly encountered in daily practice, necessitated a cesarean section. General anesthesia, instead of spinal/epidural anesthesia, was used because of the thrombocytopenia. PPH due to uterine atony occurred and rapidly consumed the residual reserve of

platelets, which could have jeopardized the development of coagulopathy. During normal pregnancy, major changes in hemostasis include increasing concentrations of most clotting factors, decreasing concentrations of some natural anticoagulants, and diminishing fibrinolytic activity. These changes create a state of hypercoagulability, thereby decreasing bleeding complications of delivery [6]. However, the most important initial factor for hemostasis at delivery is uterine contraction, which leads to the constriction of spiral arteries and stoppage of bleeding [7]. Although gestational thrombocytopenia *per se* is not the major cause of PPH, it may exacerbate the severity of bleeding complications such as diffuse intravascular coagulation.

In conclusion, platelet transfusion in patients with severe gestational thrombocytopenia or in those with high risk for postpartum hemorrhage is recommended in order to minimize the risk of a cascade of complications.

References

1. Boehlen F, Hohlfeld P, Extermann P, Perneger TV, de Moerloose P. Platelet count at term pregnancy: a reappraisal of the threshold. *Obstet Gynecol* 2000;95:29–33.
2. Fay RA, Hughes AO, Farron NT. Platelets in pregnancy: hyperdestruction in pregnancy. *Obstet Gynecol* 1983;61:238–40.
3. Shehata N, Burrows R, Kelton JG. Gestational thrombocytopenia. *Clin Obstet Gynecol* 1999;42:327–34.
4. Sainio S, Kekomaki R, Riikonen S, Teramo K. Maternal thrombocytopenia at term: a population-based study. *Acta Obstet Gynecol Scand* 2000;79:744–9.
5. Horn EH, Kean L. Thrombocytopenia and bleeding disorders. In: James DK, Steer PJ, Weiner CP, Gonik B, eds. *High Risk Pregnancy: Management Options*, 3rd edition. Philadelphia: Elsevier Saunders, 2005:901–24.
6. Burrows RF, Kelton JG. Thrombocytopenia at delivery: a prospective survey of 6715 deliveries. *Am J Obstet Gynecol* 1990;162:731–4.
7. Kam PC, Thompson SA, Liew AC. Thrombocytopenia in the parturient. *Anaesthesia* 2004;59:255–64.